ARCHIVES OF DISEASE IN CHILDHOOD

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Annotation

Head injury—abuse or accident?

'Did he fall, or has he suffered inflicted injury?' is a question faced frequently by clinicians caring for infants and toddlers with traumatic brain injury. Publicised court cases, with widely divergent medical opinions, illustrate the dilemma of distinguishing between inflicted and accidental causes, especially when there are no other signs of abuse but just an uncorroborated, alleged accident, often a fall. Although there has been resistance to diagnosing abuse there may also be over enthusiasm to do so, and although there is an increasingly prevalent opinion that short falls can never cause serious injury,[1] this, too, is still open to debate.

Causes of variability in injury

Determinants of injury severity for a given trauma mechanism such as a fall may include:

- The distance fallen.
- The nature of the surface on to which the child falls.
- Forwards or sideways protective reflexes; there is no backwards protective reflex or righting reflex.
- Whether a fall is in some way 'broken'.
- Whether the child propelled himself.
- The mass of the body and of the head.
- What proportion of the total kinetic energy is absorbed in deforming the skull, the brain or the rest of the body, and in compressing the ground; this itself may be influenced by which body part hits the ground first.
- Whether or not some kinetic energy is dissipated in causing fractures.
- Whether the contact with the ground is focal or diffuse, that is whether the fall is on to a point or on to a flat surface.
- Secondary brain injury can confuse the picture, for example hypoxic encephalopathy from an unprotected airway, or ischaemic from cerebral oedema.

Very few of these aspects have been subjected to scientific scrutiny and some of the literature relates to adults rather than children.

Falls: how far is fatal?

Short distance household falls do not normally cause serious brain injury,[2 3] but there are reports of serious injury or death in slightly longer non-household falls[4 5] and rarely in household falls.[1 6] In some of these studies a disproportionate number of deaths occurred in alleged household falls, especially when there was no corroboration of the history, suggesting that the real cause was abuse.[2 3 5 6] In some, however, extensive investigations did not prove abuse and some occurred while the child was under 'medical observation'.[6] Thirteen patients in Glasgow, including three children, died from severe cerebral injury (diffuse axonal injury) sustained in a fall from greater than their own height.[4] Four fell down a flight of stairs, one off a wall, one from a lorry; most had raised pressure, fractured skull, and cerebral contusions. Of 45 children from New York who had fallen at least 3 metres (one to six storeys), only 19 had head injury and there was no correlation between injury severity and distance fallen.[7] Various types of intracerebral haemorrhage were seen including subdural.

Altogether 317 children from San Diego with alleged falls were reviewed; falls of less than 1.2 metres were never fatal.[2] One hundred toddlers were examined, hospitalised for head injury in Philadelphia, 73 caused by falls.[3] The 34 alleged household falls (less than 1.2 metres) had a high incidence of other features strongly suggestive of inflicted as opposed to accidental injury. Some had subdural or retinal haemorrhage and two died. Two studies in California found similarly that severe injuries may occur in falls of 1.5–12 metres but never less than 1.5 metres, and death is very rare even in long falls.[5 7 8]

Fractures and extradural haemorrhage

Linear parietal skull fractures[9 10] and extradural haemorrhage[11–13] (the latter can kill) can occur in short distance falls without initial unconsciousness. A fall of just 1 metre is probably sufficient to cause a fracture in adults and in children even on to a padded surface.[14 15] Fractures are more likely to be caused by high force trauma, including abuse, if depressed, wider than 3 mm, multiple, stellate, crossing a suture line or of the base of the skull.[9 10] Fracture does not have to accompany severe brain injury, whatever the cause.[7]

Retinal haemorrhage

Retinal haemorrhage may occur alone in accidental and inflicted injury, but more often accompanies subdural haemorrhage[16] and has similar significance.[10] Trauma of increasing severity causes peripheral subhyaloid haemorrhage, then intraretinal haemorrhage, then subretinal and choroidal and vitreous haemorrhage and retinal detach-

ment, and the intracranial correlates are subdural, then subarachnoid, then intracerebral haemorrhage and diffuse brain injury.[16] A retinopathy with widespread paravenous petechiae suggests shaking injury in small infants, caused by venous hypertension as the infant's chest is gripped tightly.[10] It is most important to obtain a paediatric ophthalmology opinion to differentiate the various types of ocular injury.

Cerebral injury

Depth of coma does not necessarily define severity; children can be deeply unconscious after a minor head injury and display neurological signs such as decerebrate posturing but recover over minutes to hours, or are not unconscious initially, but develop coma later in the first day with cerebral oedema and intracranial haemorrhage. For example, four children in Toronto had a lucid interval after a minor or moderate head injury but lapsed into coma suddenly and died with cerebral contusions, cerebral oedema, and hypoxic-ischaemic encephalopathy.[17] Also, one cannot infer that the original injury was severe just because there is final, significant, neurological disability, which can result even from apparently minor injuries.[13 18]

Diffuse brain injury occurs after severe head trauma and may cause prolonged deep coma, often associated with intraventricular, subarachnoid, or intracerebral haemorrhage (contusions) or cerebral oedema and mid-line shift which all predict poor outcome.[19] Because it only occurs in traumatic injury it is thought to be caused by mechanical stretching of axons[10] in high acceleration/deceleration, high rotational strain accidents with or without impact.[20 21]

Secondary hypoxic or ischaemic injury may exacerbate traumatic brain injury and make the original trauma mechanism appear worse than the stated history, and cause preventable mortality and morbidity even in moderate head injury.[22–25]

Can minor trauma cause subdural haemorrhage?

The Japanese neurosurgical literature contains several reports of subdural haemorrhage in infants, some caused by inflicted blows, some allegedly by simple falls. A diagnostic category of infantile acute subdural haematoma was proposed, caused by minor injury in some infants with a wider than usual subdural space, usually an incidental finding on cranial computed tomography.[26 27] The wide subdural space would increase the fragility of the bridging veins, more fragile anyway in infants.[28] Critics suggested that the Japanese may be missing the shaken baby syndrome. They replied showing that inflicted injury causing subdural and retinal haemorrhage is certainly recognised in Japan but caused mostly by blows.[29] They suggested that maybe non-accidental injury was overdiagnosed in the USA, rather than underdiagnosed in Japan. Dr Anthony Raimondi, a neurosurgeon in Chicago, wrote in a comment after this paper, 'The conclusions of these authors are sound…they seriously question the tenet that coexisting subdural haematoma and retinal haemorrhage are pathognomonic of battery. Of course they are right; one cannot ascertain that a head injury is caused by battery simply because subdural haematoma and retinal haemorrhage are present…there is no way to identify unequivocally the battered child. This is true in the US and…in Japan'.

Dr Raimondi himself reported a series of subdural haemorrhages in infants and toddlers 20 years ago.[30] Both abuse and falls were identified as causes. A group in London analysed a series of subdural haemorrhage in children caused by head trauma concluding that some patients are at higher risk of subdural caused by minor injury, and also that non-accidental injury is possibly overdiagnosed.[31]

Subdural haematoma contributing to neurological deterioration, cerebral oedema or death has been described in adults and children after only moderate or trivial head injury.[13 18 32] Asymptomatic brain contusions and subdural haemorrhage have also been identified on computed tomography or magnetic resonance imaging in adults.[33]

How much acceleration is needed to cause brain injury?

A group from Philadelphia suggested that 'whiplash shaking' alone may not after all cause severe brain injury,[21] despite two decades of evidence to the contrary.[10] First, they found that there was rarely actually any history of shaking, other causes with impact being more likely. Secondly, a model baby's skull, when the doll was shaken vigorously, sustained linear acceleration, measured by an implanted accelerometer, of only 9 G, nothing like what they claimed was needed to cause concussion, let alone subdural haemorrhage (about 285 G) or even diffuse axonal injury, although this was based on extrapolation from experiments where acceleration injury was inflicted on anaesthetised adult monkeys.[34] Acceleration was 428 G when the model head impacted on a surface during a shake. Other mechanisms have been proposed for shaking alone causing severe injury in infants, including trauma to the large vessels of the neck, and trauma to the cervical spinal cord.[35]

In a study, by an Australian road accident research unit, of fatally injured pedestrians hit by cars, it was estimated (it is impossible to measure acceleration in real accidents) that in adults peak linear accelerations of as little as 150 G could cause brain injury, with contusions on computed tomography, leading to death, and even less than 150 G if the impact was to the side of the head.[36]

Another group has reported three toddlers who all died from inflicted injury, presumably a blow to the side of the head, with bruising in the pinna, ipsilateral subdural haemorrhage and brain swelling, and retinal haemorrhage.[37] The acceleration which they calculated would be needed to produce this (extrapolated from minimal adult data) was much less than the Philadelphia group suggested.

To gain some insight into these disparate findings we turn to a mechanical engineering discipline—the study of dynamics.

Dynamics of falls and blows

It has been said that packed earth can still cause 200 G deceleration after a 1.2 metre fall.[38] This can be calculated from force/compression data, measured for a given object and ground type by a compression dynamometer. As an example, the following figures were calculated from actual data obtained for a 1.5 kg, 15 cm sphere (equivalent of a 12 kg toddler's head) indenting packed earth, for a fall of 1.5 metres: velocity, 5.4 m/sec at impact; kinetic energy, 21.9 Joules at impact; deceleration, mean 81 G, peak 238 G; force, peak 3500 Newtons; distance indented, 18.6 mm; stopping time 4.8 msec, assuming all kinetic energy is expended as work of compression (C Wainwright, personal communication). These figures are for the skull—they cannot be calculated for the brain. Deceleration is greater and indentation less for smaller objects, for a harder surface, and if (as must be the case) some of the kinetic energy on impact is absorbed into the head as deformational strain in the skull[39] or brain. Deceleration is greater for a higher fall—doubling the fall less than doubles velocity but more than doubles peak deceleration. Kinetic energy of a falling object is proportional to mass and to distance fallen, and independent of ground type. At impact some of this energy is dissipated as work of compressing the

ground as the object decelerates. Soft ground may be less injurious than hard, not necessarily because acceleration is less (it operates over a longer time) but because more kinetic energy is absorbed by the surface, less by the head.

Acceleration magnitude probably matters less than how much kinetic energy is absorbed into the brain and subdural bridging veins, and dissipated in tearing veins and intracerebral vessels, and in stretching neurones (kinetic energy is proportional to the integral of acceleration over distance). The time course and magnitude of these energy changes in the brain cannot be measured or calculated.

In a blow acceleration is lesser and of longer duration than that for a fall, but in reverse with acceleration and force maximum at the moment of impact.[37] Also, in experiments on model heads, blows and falls of the same force caused different intracranial pressure waves, possibly accounting for falls causing more 'contre-coup' injury, and blows 'coup' injury in the brain.[40]

Linear and rotational motion

Why might some lesser trauma, with less energy, cause more severe injury? Acceleration of the head, with or without impact, will cause dynamic relative motion of the brain within the skull. For pure linear or translational motion brain and skull move in the same direction, and brain deflection is small; it will strike the inside of the skull, possibly causing coup or contre-coup contusion. Bridging veins will be stretched only a little and will not arrest motion.

The Philadelphia group emphasised the need for rotational rather than linear acceleration or deceleration to cause serious brain injury.[20 21 34] However, their axis of rotation is external to the skull, at the lower cervical spine, the movement being in the coronal plane (for example blow to side of head), or in the sagittal plane (for example whiplash injury, blow to back of head, or fall backwards). The skull and entire brain both travel in an arc in the same direction, ie their relative motion is still largely linear (tangential) with only a minor angular component, if any.

True rotation of the brain within the skull will occur when the centre of rotation is internal to the skull (for example axial as in a blow to the side of the jaw, or possibly in an oblique strike by a car or in a rolling fall), the motion transferred from skull to brain via shear rather than compressive forces. The bridging veins are potentially stretched more and there is likely to be less damping by cerebrospinal fluid, so kinetic energy stored in the brain may be dissipated in tearing these vessels. Similar shearing forces may operate within the brain causing diffuse brain injury. Lesser true rotational forces are needed than linear forces to cause such injury. True rotation might be rare in simple falls but explain rare cases of severe injury.

Why might shaking babies cause severe injury despite low acceleration and motion which is largely linear? A possible reason is that the motion is repetitive (maybe 4 Hz[21]). If this or a harmonic is close to the resonant frequency (which might be as low as 10 Hz), there is potential for brain movement of high amplitude. Secondly, there may be axial rotation of the baby's head on the neck during violent backwards/forwards shaking. Thirdly, it has been shown that the spinal cord may be injured, in babies shaken with no evidence of impact, with extradural, intradural, and intracord haemorrhage within the spinal canal.[35]

Because of resonance and damping the motion of the brain is time dependent and a very short, high force impact could cause less injury, or different type of injury, than a lesser force applied over a longer period.[34] There is some evidence, for example, that rapid onset acceleration tends to cause subdural haemorrhage, whereas a softer impact causes diffuse brain injury. Blows to the side of the head are more damaging than to the occiput,[20 36 37] possibly because resonance and damping may differ in the two directions and possibly because a blow to the side of the head induces true rotation. Conversely, falls on to the occiput may be more damaging because there is no protective reflex as in a sideways or forwards fall.

Conclusion

Small infants rarely sustain serious injury from accidents in the home and any brain injury with subdural and retinal haemorrhage should raise suspicions of abuse. Babies can, however, be dropped accidentally or fall from changing tables and sustain linear fractures and epidural haemorrhages. We must be more cautious in toddlers who are mobile, can climb and have unwitnessed accidents. In the absence of clear signs of abuse we cannot jump to the conclusion that injury is non-accidental just because there is brain injury or subdural haemorrhage, especially if the alleged fall height is greater than in 'household' falls.

Measurement of forces applied to the skull does not permit calculation of forces imposed on the brain, or of how much force (or acceleration, or kinetic energy) and duration of that force are needed to produce various types and severity of injury. There are too many variables and unknowns to allow a categoric statement that a certain fall did or did not injure a child. A detailed medical and social history may raise suspicion of abuse, but the medical appraisal may not be conclusive about whether an injury is compatible with the stated history. Evidence given in court must be unbiased and factual; we must not allow our rightful abhorrence of abuse in all its forms to to blind us to the precept that a person is innocent until proved guilty.

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Commentary

'Science is the father of knowledge, opinion breeds only ignorance'. Hippocrates stressed the importance of using evidence in achieving diagnosis. There are few diagnostic dilemmas more challenging in medicine than the separation of accidental from non-accidental injuries. There is no room for error, families wrongly accused lose more than their child, while a child returned in error to an abusing home may lose its life. The brain is an infant's most valuable and most vulnerable organ. Dr Wilkins has crystallised the diagnostic problems presented by infant brain injury and summarised many of the current controversies. He has also identified the increasing concern among legal and forensic colleagues about a small number of over confident diagnoses where evidence is lacking. He has summarised the bulk of the available evidence and highlighted how other disciplines can facilitate resolution.

The controversies arise mainly because of the impossibility of carrying out objective experimental injuries to a child's brain. The bulk of the available information is abstracted from reported injuries, animal experiments, and theoretical models. Speculative extrapolations from these data may lead to difficulty. Such uncertainties apply to other areas of medicine but few, if any, are subject to such intense public scrutiny.

The effects of violence towards the vulnerable are well recognised and documented. In the majority of cases there is little doubt and little diagnostic difficulty. When there are multiple injuries of different ages in different sites with inconsistent history, there can be no plausible alternative explanation to abuse. The difficulties that currently arise in a minority of cases should not distract or detract from the needs to protect other children.

Human skull fractures have been created in mortuary experiments, the known facts are:

- 80% of stillbirths dropped 18 inches onto a paved floor sustained skull fracture.[1]
- Infant cadavers aged 2 to 8 months dropped 82 cm onto concrete, foam back linoleum, or thin carpet all sustained skull fractures,[2] interposing 8 cm of camel blanket reduced the fracture rate to 16% and 2 cm of rubber reduced it to 10%.[3]
- Adult cadaver heads dropped four feet onto a hard surface (terminal velocity 10 mph) all sustained skull fractures.[4]

These figures are at variance with reported observational studies of the hundreds of children who fall in domestic and hospital accidents but do not sustain serious injury.[5 6] These latter studies have led to confident extrapolations such as 'skull fracture in children is impossible with a fall less than 10 feet'. This apparent conflict can be reconciled by the uncertainty in the observational studies of the true free fall, protective reflex, and landing surface. Among 207 observed hospital falls there were only two fractures, one infant fractured the skull with an estimated impact of 39 Newtons (N), the other had an estimated impact of 103 N yet only fractured the clavicle[7]; many of the other children had impacts of greater than 40 N but did not sustain fractures.

Acute subdural haematoma and axonal shearing injuries have been created by shaking anaesthetised primates.[8] It was impossible to replicate the necessary energy transfers in an infant doll model by shaking alone, additional striking or impact were needed; suggesting that we should consider a 'shaken impact syndrome'. Possible alternative explanations are that the primate experiments are more relevant to adult brain injury or that the model was flawed. The model was a life size infant doll's head *packed tight* with wet cotton waste. All models to date have ignored the relatively large cerebrospinal fluid (CSF) space in infants (up to 1 cm), the stabilising influence of falx cerebri and tentorium cerebelli, damping of brain movement by CSF, a possible water hammer effect as CSF moves in the opposite direction to the rotating brain, or a 'whipcrack' effect of inertial movements of the brain inside the rapidly rotating skull.

The first model to successfully explain brain injury in the absence of skull or scalp injury was taken from traffic accident work[9]: 'A few spoonfuls of dessicated coconut suspended in liquid paraffin in a round bottom flask can be more readily set in motion and swirled for longer after a few seconds of gentle shaking than after the hardest blow that could be delivered without breaking the flask'. This shaking also explained metaphyseal fractures to flailing limbs, with gripping causing posterior rib fractures or bruising. It has served us well but may need to be revised as knowledge advances.

Diagnostic errors have been made in the past when confidently stated dogma has been repeated and built upon. Increasing experience may merely allow the same mistake to be made with increasing confidence. The controversy between Japanese and American experts summarised by Dr Wilkins has also now involved British neurosurgeons.[10] The possibility of some racial predisposition to acute infantile subdural haematoma in Japanese and Afro-Caribbean children has not been scientifically excluded.[10–12] There is no other recognised racial predelection for abuse, it may be part of the human condition. There is also no recognised gender predisposition to physical abuse yet all studies of infantile acute subdural haematoma show a striking male preponderance: just like

accidents. The age distribution peaking between the ages of early rolling to confident walking is epidemiologically consistent with an accidental aetiology. Many of us have exhibited logical inconsistency by believing parents who say they have shaken their baby but disbelieving those who deny it, and we believe parents who describe a fall where no significant injury occurs but disbelieve others when injuries are found. Religious logic is a different process from scientific thought.

Such disbelief is of course correct when there are multiple injuries of different ages. There is now a recognised association between magnitude of energy transfer and severity of injury, increasing insults result in increasing injuries ranging from scalp contusion through linear parietal fracture, stellate skull fracture, basal skull fracture, subdural haematoma, retinal haemorrhage, axonal injury, and death. Advances in imaging now enable segregation of different injuries allowing more directed forensic questioning. Comparisons of changing images with time after traffic accidents has allowed more confident identification of the likely time of insult which can challenge offered histories.[13] Axonal shearing injuries require high levels of acceleration/deceleration[8] and can now be identified by computed tomography, magnetic resonance imaging, and high resolution ultrasound scanning.[14–16] Ultrasound scanning can be done at the cot side in the intensive care unit. Incorporation of such data are part of the continued collaborative clinical work which is the hallmark of forensic paediatrics.

Much of the advance in our understanding of brain injury in child abuse has come from extrapolations of traffic accident research. Current work may offer considerable hope for reducing the levels of morbidity and/or preventing mortality in those who suffer abuse. There is a need for further and continued preventive efforts, little has changed since Guthkelch stated 'many parents consider a good shaking to be more socially acceptable and physically less dangerous than a blow to the head'.[9]

Child protection is not solely dependent upon the diagnostic clinician. The integration of social data and forensic interviewing clarifies many of the difficult cases. In a minority there may remain a residual doubt. In these cases, over enthusiastic extrapolation from speculative theoretical studies may result in an over confident opinion that can, in the long term, serve only to harm. Historical studies of thousands of children with brain injury where more than half were attributed to unwitnessed accidental falls and none to abuse even where there was subdural haematoma and retinal haemorrhage[17 18] may not have always been correct. It is however not scientifically certain that such a constellation of injury is always due to abuse; the truth may lie between these extremes.

Social legislation endeavours to secure the best solution for a child in an imperfect world; there is no place for revenge in justice. Given the infinite variety of ways in which children can be injured and respond to injury, each case must be considered on its own merits. We must not fall prey to the primary scientific fallacy 'where the facts fit the theory accept them; where they don't, change the facts not the theory'. In the absence of definitive physical signs we may need to profess fallibility. Humility avoids hubris. False diagnoses not only harm children and their families but also devalue the profession. The poet Piet Hein expressed this well: 'My faith in doctors is quite immense, my only fear is their pretence of some divine omniscience'.

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